In The Claims

Please amend the claims by replacing all prior versions of the claims pursuant to 37 C.F.R. §1.121 as modified by 68 Fed. Reg. 38611 (June 30, 2003) as follows:

1. (Currently Amended) A method for treating or preventing stroke in a human subject susceptible to ~~intracranial~~ intracerebral hemorrhaging, comprising administering to the human subject an effective amount of a CD39 polypeptide comprising consecutive amino acids the sequence of which is set forth in SEQ ID NO:1 or an active polypeptide fragment thereof so as to inhibit adenosine diphosphate-mediated platelet aggregation by increasing adenosine diphosphate catabolism without increasing incidence of intracerebral hemorrhage in the human subject.

2. (Previously Presented) The method of claim 1, wherein the active polypeptide fragment of CD39 polypeptide is administered and is a mutated or a truncated form of the CD39 polypeptide.

3. (Currently Canceled)

4. (Currently Canceled).

5. (Currently Canceled).

6. (Currently Canceled).

7. (Currently Amended) The method of claim 1, wherein the active polypeptide fragment of the CD39 polypeptide comprises consecutive amino acids the sequence of which is identical to the sequence of amino acid residues ~~about~~ 20-80

~~amino acid residues~~ of SEQ ID NO:1 ~~which mimics the active site of CD39~~.

8. (Currently Canceled)

9. (Previously Presented) The method of claim 1, wherein the administration of the CD39 polypeptide or the active polypeptide fragment thereof is effected at the onset of stroke in the human subject.

10. (Previously Presented) The method of claim 1, wherein the administration of the CD39 polypeptide or the active polypeptide fragment thereof is effected prior to stroke onset in the human subject.

11. (Previously Presented) The method of claim 1, wherein the administration of the CD39 polypeptide or the active polypeptide fragment thereof is effected after stroke onset in the human subject.

12. (Currently Amended) The method of claim 1, wherein the CD39 polypeptide or the ~~its~~ active polypeptide fragment is administered in a dosage of 1-20 mg/kg of the subject's body weight.

13. (Currently Amended) The method of claim 1, wherein the CD39 polypeptide or the ~~its~~ active polypeptide fragment is administered in a dosage of 4-8 mg/kg of the subject's body weight.

14-16. (Currently Canceled)

17. (Currently Amended) A method for ~~determining whether~~ testing a compound ~~inhibits platelet aggregation or leukocyte~~

~~accumulation by increasing ADP catabolism and does not increase incidence of intracerebral hemorrhage, so as to treat or prevent thrombotic or ischemic disorder in a subject,~~ comprising:

(a) administering the compound ~~to an animal~~, which compound increases adenosine diphosphate catabolism, to a CD39-deficient mouse ~~is a~~ model for ~~the~~ a thrombotic ~~or ischemic~~ disorder~~, before,~~ concurrently with or after step (b);

(b) inducing the thrombotic ~~or ischemic~~ disorder in the mouse ~~animal~~;

(c) measuring stroke ~~the thrombotic or ischemic disorder~~ outcome and ~~the~~ incidence of intracerebral hemorrhage in the mouse ~~animal~~;

(d) measuring platelet deposition ~~and/or fibrin deposition and/or accumulation of leukocytes~~ in ischemic tissue in the mouse ~~animal~~; and

(e) comparing the ~~thrombotic or ischemic disorder~~ stroke outcome and the incidence of intracerebral hemorrhage and the platelet ~~and/or fibrin~~ deposition ~~and/or accumulation of leukocytes~~ in the presence of the compound with ~~in~~ the incidence of intracerebral hemorrhage and platelet deposition in the absence of the compound, wherein a decrease in platelet deposition but no increase in the incidence of intracerebral hemorrhage indicates that ~~so as to determine whether~~ the compound is capable of treating ~~or preventing~~ the thrombotic ~~or ischemic~~ disorder in the subject ~~without increasing the incidence of intracerebral hemorrhage~~.

18. (Currently Amended) The method of claim 17, wherein ~~the animal model comprises CD39-deficient mice and wherein~~ the thrombotic ~~or ischemic disorders are~~ disorder is induced by administering ~~an~~ a platelet agonist to said mice.

19. (Previously Presented) The method of claim 17, wherein the stroke outcome is determined from measurements of platelet deposition, bleeding time and infarction volume.

20. (Original) The method of claim 17, wherein the compound can be administered orally or by injection.

21. (Currently Canceled).

22. (Currently Amended) The method of claim 17, wherein the administration of the compound is ~~prior to stroke onset in the animal~~ before step (b).

23. (Currently Amended) The method of claim 17, wherein the administration of the compound ~~occurs at the onset of stroke in the animal~~ is concurrent with step (b).

24. (Currently Amended) The method of claim 17, wherein the administration of the compound ~~occurs after stroke onset in the animal~~ is after step (b).

25-26. (Currently Canceled)

27. (Original) A method for treating an ischemic disorder in a subject which comprises administering to the subject a CD39 polypeptide (SEQ ID NO. :1) or an active fragment there of which inhibits ADP or ATP mediated platelet aggregation or leukocyte accumulation so as to treat the ischemic disorder in the subject.

28. (Original) The method of claim 27, wherein the leukocyte is a white blood cell, a neutrophil, a monocyte or a platelet.

29. (Original) The method of claim 27, wherein the subject is a mammal.

30. (Original) The method of claim 27, wherein the mammal is a human.

31. (Original) The method of claim 29, wherein the ischemic disorder comprises a peripheral vascular disorder, a pulmonary embolus, a venous thrombosis, a myocardial, infarction, a transient ischemic attack, unstable angina, a reversible ischemic neurological deficit, sickle cell anemia or a stroke disorder.

32. (Original) The method of claim 27, wherein the subject is undergoing heart surgery, lung surgery, spinal surgery, brain surgery, vascular surgery, abdominal surgery, or organ transplantation surgery.

33. (Original) The method of claim 32, wherein the organ transplantation surgery comprises heart, lung, pancreas or liver transplantation surgery.

34. (New) A method for treating stroke in a human subject susceptible to intracerebral hemorrhaging, comprising administering to the human subject an amount of a CD39 polypeptide, which CD39 polypeptide comprises consecutive amino acids having the sequence shown in SEQ ID NO:1, effective to inhibit adenosine diphosphate-mediated platelet aggregation by increasing adenosine diphosphate catabolism without increasing incidence of intracerebral hemorrhage in the human subject so as to thereby treat stroke in the human subject.

35. (New) A method for treating stroke in a human subject susceptible to intracerebral hemorrhaging, comprising administering to the human subject an amount of an active fragment of a CD39 polypeptide comprising consecutive amino acids, the sequence of which CD39 polypeptide is set forth in SEQ ID NO:1, effective to inhibit adenosine diphosphate-mediated platelet aggregation by increasing adenosine diphosphate catabolism without increasing incidence of intracerebral hemorrhage in the human subject, wherein the active fragment is further characterized in that it decreases platelet deposition but does not increase the incidence of intracerebral hemorrhage when the active fragment is administered to a CD39-deficient mouse model for a thrombotic disorder after inducing the thrombotic disorder in the mouse, and stroke outcome, incidence of intracerebral hemorrhage, and platelet deposition in the mouse are measured.

36. (New) A method for treating stroke in a human subject susceptible to intracerebral hemorrhaging, comprising administering to the human subject an amount of a deletion mutant, substitution mutant, or insertion mutant of a CD39 polypeptide comprising consecutive amino acids, the sequence of which CD39 polypeptide is set forth in SEQ ID NO:2, effective to inhibit adenosine diphosphate-mediated platelet aggregation by increasing adenosine diphosphate catabolism without increasing incidence of intracerebral hemorrhage in the human subject, wherein the deletion mutant, substitution mutant, or insertion mutant is further characterized in that it decreases platelet deposition but does not increase the incidence of intracerebral hemorrhage when the deletion mutant, substitution mutant, or insertion mutant is administered to a CD39-deficient mouse model for a thrombotic disorder after inducing the thrombotic disorder

in the mouse, and stroke outcome, incidence of intracerebral hemorrhage, and platelet deposition in the mouse are measured.